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Obstetric and Neonatal Adversities, Parity, and Tourette Syndrome: A Nationwide Registry

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Objective To determine the relationships between parity, obstetric adversities, neonatal factors, and Tourette syndrome in a large nationwide cohort.

Study design This nationwide, register-based, nested case-control study identified all children diagnosed with Tourette syndrome born between 1991 and 2010 from the Finnish Hospital Discharge Register (n = 767). Each case was matched to 4 controls. Information on parity, obstetric, and neonatal factors was obtained from the Finnish Medical Birth Register. Conditional logistic regression was used to determine the relationship between parity, obstetric, and neonatal factors, and Tourette syndrome.

Results Nulliparity was associated with increased odds for Tourette syndrome (OR 1.7, 95% CI 1.4-2.2), and 3 or more previous births was associated with decreased odds for Tourette syndrome (OR 0.5, 95% CI 0.3-0.9) compared with parity 1-2. Birth weight 4000-4499 g was associated with decreased odds for Tourette syndrome (OR 0.7, 95% CI 0.5-0.9). Low birth weight, gestational age, weight for gestational age, Apgar score at 1 minute, induced labor, birth type or presentation, neonatal treatment, or maternal blood pressure were not associated with Tourette syndrome.

Conclusions Increasing parity and high birth weight are associated with decreased odds for Tourette syndrome. *(J Pediatr 2016;171:213-9)*.

See editorial, p 17

Tourette syndrome is a childhood onset neurodevelopmental disorder characterized by multiple motor and 1 or more vocal tics that have been present for more than a year.[1] The complex etiology of Tourette syndrome is likely to involve multifactorial genetic and environmental influences,[2-4] although underlying pathophysiological mechanisms and the role of potential gene-environmental interactions are as yet unclear.

Both obstetric and neonatal factors have been associated with neurodevelopmental disorders, such as attention deficit hyperactivity disorder (ADHD) and autism spectrum disorder (ASD).[5-11] A growing body of evidence suggests that parity, particularly being the first born, is associated with an increased risk of autism.[12-14] Most previous studies on the association between obstetric and neonatal factors and Tourette syndrome were retrospective in design and conducted on clinical samples.[15-23] Low birth weight,[15,16] complications during pregnancy,[17] and Apgar scores at 5 minutes[18] have been reported to be associated with Tourette syndrome[16,18] or tic severity.[15,17] These findings, however, have not been replicated or have been inconsistent.[15,18-21] Two population-based studies have examined the association between perinatal factors and Tourette syndrome: one from Sweden[23] and one derived from the Avon Longitudinal Study of Parents and Children (ALSPAC) birth cohort.[24] The Swedish study included only 25 children with Tourette syndrome and examined the association between combined pre- and perinatal optimality score and Tourette syndrome. No specific perinatal factors were evaluated.[23] The ALSPAC birth cohort study included 50 children with Tourette syndrome; no associations were found between birth weight, prematurity, delivery complications, Apgar score, or maternal blood pressure and Tourette syndrome.[24] However, a negative, statistically significant (OR 0.7 95% CI 0.5-0.97) association was found between parity (second or later born vs first born) and Tourette syndrome/chronic tic disorder.[24] Of note, Tourette syndrome and chronic tic disorder diagnoses in the ALSPAC cohort were based

ADHD	Attention deficit hyperactivity disorder
ALSPAC	Avon Longitudinal Study of Parents and Children
ASD	Autism spectrum disorder
FHDR	Finnish Hospital Discharge Register
FMBR	Finnish Medical Birth Register
ICD	*International Classification of Diseases*
ICD-10	ICD Tenth Revision
ICD-9	ICD Ninth Revision
SES	Socioeconomic status

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on questionnaires completed by mothers.[24,25] Specific obstetric and neonatal adversities are relatively uncommon. Therefore, the low statistical power because of small sample sizes used by these studies[23,24] may have resulted in failure to demonstrate subtle but important associations between obstetric and neonatal factors and Tourette syndrome. A design based on using national health register data overcomes many limitations (eg, retrospectively collected data, small sample sizes, geographically restricted area, and lack of healthcare professional assessments) of the previous clinical and population-based studies.

The present study is a nationwide register-based study examining the relationships between several obstetric adversities, perinatal factors, parity, and Tourette syndrome. The aim of the study was to examine if birth weight, gestational age, weight for gestational age, Apgar score at 1 minute, induced labor, birth presentation, birth type, maternal blood pressure, neonatal treatment, or parity are associated with Tourette syndrome after adjusting for other parental background variables.

Methods

This nested case-control study used data derived from 3 Finnish national registries. The sampling frame included all children born between January 1, 1991, and December 31, 2010, in Finland (n = 1 199 112). Children diagnosed with Tourette syndrome (n = 767) during the same time period were identified from the Finnish Hospital Discharge Register (FHDR). The characteristics of the sample have been described previously.[26] Each case was matched to 4 controls, identified from the Central Population Register, by sex, date of birth, and place of birth. Personal identity codes were used to link the data collected from the FHDR, the Finnish Medical Birth Register (FMBR), and the Central Population Register used in the study. The study was authorized by the Ministry of Social Affairs and Health (STM/1528/2007) and the National Institute of Health and Welfare with approval from the ethics committee of the hospital district of Southwest Finland.

The FHDR contains all somatic and psychiatric diagnoses given in hospitals in Finland, including inpatient wards, military, health center, and prison wards, and private hospitals from 1969 onward and public hospital outpatients from 1998 onward. Diagnoses are based on the *International Classification of Diseases* (ICD) (ICD Ninth Revision [ICD-9] from 1987-1995 and ICD Tenth Revision [ICD-10] from 1996 onward). FHDR data have been shown to be of good quality,[27] and more specifically, Tourette syndrome diagnoses in the FHDR have been validated by chart reviews and telephone interviews using the Yale Global Tic Severity scale.[26] The Central Population Register contains information about Finnish citizens and foreign citizens residing permanently in Finland, and includes name, personal identity code, address, citizenship, native language, family relations, and dates of emigration, immigration, birth, and death. The FMBR, established in 1987 and maintained by the National Institute of Health and Welfare, includes detailed comprehensive data on maternal background, pregnancies, and the perinatal period up to 7 days of age.

Children with diagnosed Tourette syndrome (ICD-10: F95.2, ICD-9: 3072D) without comorbid severe or profound intellectual disability (ICD-10: F72, F73, ICD-9: 3181, 3182) were identified from the FHDR (n = 767). Children born from twin (n = 24) or triple (n = 1) pregnancies were excluded. Data on perinatal factors were available for 738 of the remaining 742 children (99.5%). There were 207 Tourette syndrome cases with comorbid hyperkinetic disorder, 120 cases with comorbid ASD, and 58 cases with comorbid obsessive compulsive disorder.

The controls, defined as children without any tic disorder or severe or profound intellectual disability, were identified from the Central Population Register. Controls were excluded if they had emigrated from Finland (n = 9), had died before the case was diagnosed (n = 12), were born from multiple pregnancies (n = 87), or if their case was born from a multiple pregnancy (n = 98). The data on perinatal factors was available on 2810 of the remaining 2825 controls (99.5%).

Data on Exposures

Data on parity, obstetric, and neonatal factors were derived from the FMBR. The studied obstetric and neonatal factors were: (1) birth weight classified as <1500, 1500-2499, 2500-3999, 4000-4499, and ≥4500 g; (2) gestational age classified as <32, 32-37, 38-41, and ≥42 weeks; and (3) weight for gestational age as small for gestational age (<−2 SD), appropriate for gestational age (−2 SD to 2 SD), and large for gestational age (>2 SD). Weight for gestational age was calculated according to national sex-specific weight distribution standards at a given gestational age among children born between 1996 and 2008.[28] Apgar scores at 1 minute were classified as 9-10, 7-8, and 0-6. Neonatal treatment was classified as either normal follow-up or monitoring either in a maternal postpartum department or a neonatal intensive care unit. Birth presentation classified as cephalic, breech, or other. Birth type was classified as vaginal cephalic, vacuum extractor or forceps, or vaginal breech, planned cesarean, other cesarean including urgent and emergency cesarean, and unknown. Induced labor was classified as either yes or no. Maternal high blood pressure that required hospitalization was either yes or no. Parity was classified as 0, 1-2, and 3 or more previous births.

Covariates

Five parental factors that showed a trend of association ($P < .10$ calculated using Pearson χ^2 test) with Tourette syndrome in this sample and at least one of the perinatal factors in the literature[29-34] were included in the analyses as covariates. These covariates were maternal age ($P = .003$), maternal psychiatric history ($P < .001$), maternal socioeconomic status (SES) ($P = .001$), paternal age ($P = .001$), and paternal psychiatric history ($P < .001$). In addition, number of the siblings in the sibship ($P = .006$) was added as a covariate into final

model. In additional analyses, prenatal maternal smoking (yes/no) ($P = .007$) was also added as a covariate into final model. Maternal age was classified as <20, 20-24, 25-34, 35-39, and ≥40 years. Maternal SES, based on occupation during pregnancy, was derived from the FMBR and categorized upper white collar, lower white collar, blue collar, or other. Maternal lifetime psychiatric history, derived from the FHDR, was defined as a psychiatric diagnosis given in the specialized healthcare and classified as yes or no. "Yes" included any mental disorder corresponding to diagnoses F10-F99, excluding intellectual disability F70-F79 (ICD-10)[1]; 291-316; organic psychiatric conditions 293-294 and 310 (ICD-9), and 291-308; organic psychiatric conditions 292-294 (except psychiatric disorder because of intoxication 294.30); and conditions because of sexuality 302 (ICD, Eighth Revision). Data on maternal smoking, derived from the FMBR, were categorized as either yes or no. Number of children in the sibship included the siblings delivered by the same mother and was classified as 1, 2, 3, and 4 or more.

Data Analyses

Conditional logistic regression analysis was used to examine the association between neonatal factors, obstetric factors, parity and Tourette syndrome. The ORs and 95% CIs were calculated with a statistical significance limit of $P < .05$. First, the unadjusted ORs with 95% CIs were calculated separately for every factor and Tourette syndrome. Second, the association between every factor and Tourette syndrome was evaluated adjusting for the 5 parental covariates previously noted. Subsequently, based on the results of the adjusted models, a final model was created including the risk factors significantly associated with Tourette syndrome, the 5 parental covariates, number of the siblings in the sibship, and gestational age. In additional analyses, prenatal maternal smoking was added as a covariate in the final model. Statistical analyses were performed with SAS statistical software (v 9.4; SAS Institute Inc, Cary, North Carolina).

Results

Table I shows the frequencies of examined factors among cases and controls. The unadjusted and adjusted ORs for the obstetric factors, neonatal factors, parity, and Tourette syndrome are also shown in Table I. In the adjusted analyses, statistically significant associations were found between parity, birth weight, birth type, and Tourette syndrome, respectively. Nulliparity was associated with increased odds for Tourette syndrome, and furthermore, 3 or more previous births were associated with decreased odds for Tourette syndrome compared with 1-2 previous births. Birth weight 4000-4499 g, compared with 2500-3999 g, was associated with decreased odds for Tourette syndrome and birth type (vacuum extractor, forceps, or vaginal breech) was associated with increased odds for Tourette syndrome. In the adjusted analyses, no statistically significant associations were found between gestational age, weight for gestational age, Apgar at 1 minute, induced labor, neonatal treatment, or maternal blood pressure, and Tourette syndrome.

Table II shows the final model including parity, birth weight, and birth type and covariates: maternal age, maternal psychiatric history, maternal SES, paternal age, paternal psychiatric history, gestational age, and number of the siblings in the sibship. Parity 3 or more previous births and birth weight 4000-4499 g were associated with decreased odds for Tourette syndrome. Nulliparity remained to be associated with increased odds for Tourette syndrome. In the final model, there was no statistically significant association between birth type and Tourette syndrome. There were no significant differences in the results when prenatal maternal smoking was added as a covariate into final model in the additional analyses.

Discussion

This large nationwide register-based study reports the relationship between parity, obstetric adversities, neonatal factors, and Tourette syndrome. Both increasing parity and high birth weight were significantly associated with decreased odds for Tourette syndrome. In contrast with previous findings related to other neuropsychiatric disorders, such as ASD and ADHD, Tourette syndrome was not associated with low birth weight, prematurity, or other obstetric or neonatal adversities.

Nulliparity was associated with increased odds for Tourette syndrome. In addition, the odds for Tourette syndrome decreased with increasing parity: parity greater than 2 was associated with lower odds of Tourette syndrome than parity of 1 or 2. Nulliparity has been associated with increased risk for autism,[12-14] ADHD,[35,36] schizophrenia,[37] and other mental health problems,[36,38] although these studies are not univocal.[39-42] The mechanisms underlying the association between nulliparity and adverse neurodevelopmental outcomes remain unknown. Given the expected multifactorial etiology for Tourette syndrome, it seems reasonable to consider both biological and behavioral mechanisms whereby the experience of one pregnancy changes fetal exposure and/or parental behavior in the next pregnancy in early infancy. Previously suggested biological explanations include the hygiene hypothesis,[12,13] and exposure to higher lipophilic chemicals during the first pregnancy.[12,13] The hygiene hypothesis suggests that lack of early infectious disease exposure leads to an autoimmune susceptibility among first born children and increased exposure to infections among the later born children would rather be protective.[43] This mechanism could be of relevance because abnormal regulation of the immune system could be associated with Tourette syndrome.[44] A fetus in an earlier pregnancy is exposed to higher concentrations of lipophilic chemicals than a fetus in a later pregnancy,[45] and some of these chemicals have been associated with adverse neurodevelopmental outcomes.[46] Altered maternal behavior relative to parity might also lead to different toxic exposures depending on birth order. For instance, binge drinking and cannabis use during pregnancy

Table I. The associations between neonatal factors, obstetric factors, parity, and Tourette syndrome

	Cases	Controls	Unadjusted		Adjusted model*	
	n (%)	n (%)	OR (95% CI)	*P*	OR (95% CI)	*P*
Birth weight (g)						
<1500	6 (0.8)	16 (0.6)	1.3 (0.5-3.4)	.566	1.1 (0.4-3.0)	.847
1500-2499	26 (3.5)	68 (2.4)	1.4 (0.8-2.2)	.204	1.3 (0.7-2.1)	.403
2500-3999	588 (79.7)	2074 (73.8)	1.0		1.0	
4000-4499	101 (13.7)	527 (18.8)	0.7 (0.5-0.8)	.001	0.7 (0.5-0.9)	.002
≥4500	17 (2.3)	125 (4.5)	0.5 (0.3-0.8)	.005	0.6 (0.3-1.1)	.084
Gestational age (wk)						
≤31	8 (1.1)	14 (0.5)	2.2 (0.9-5.3)	.074	1.8 (0.7-4.7)	.264
32-37	76 (10.3)	248 (8.8)	1.2 (0.9-1.6)	.207	1.2 (0.9-1.6)	.312
38-41	624 (84.6)	2421 (86.2)	1.0			
≥42	30 (4.1)	127 (4.5)	0.9 (0.6-1.4)	.684	0.9 (0.6-1.5)	.788
Weight for gestational age						
SGA	21 (2.9)	80 (2.9)	1.0 (0.6-1.6)	.998	1.0 (0.5-1.7)	.913
AGA	702 (95.1)	2628 (93.5)	1.0			
LGA	15 (2.0)	102 (3.6)	0.5 (0.3-0.96)	.034	0.7 (0.4-1.2)	.171
Apgar at 1 min						
9-10	547 (74.1)	2147 (76.4)	1.0		1.0	
7-8	152 (20.6)	561 (20.0)	1.1 (0.9-1.3)	.549	1.0 (0.8-1.3)	.828
0-6	39 (5.3)	102 (3.6)	1.5 (1.01-2.2)	.044	1.5 (0.98-2.3)	.062
Induced labor						
No	617 (83.6)	2360 (84.0)	1.0		1.0	
Yes	121 (16.4)	571 (16.1)	1.0 (0.8-1.3)	.747	1.1 (0.9-1.4)	.408
Birth type						
Vaginal cephalic	547 (74.1)	2173 (77.3)	1.0		1.0	
Vacuum extractor/forceps/vaginal breech	63 (8.5)	178 (6.3)	1.4 (1.04-1.9)	.028	1.5 (1.1-2.1)	.020
Planned cesarean	58 (7.9)	220 (7.8)	1.0 (0.8-1.4)	.792	1.0 (0.7-1.4)	.982
Emergency/urgent cesarean	70 (9.5)	233 (8.3)	1.2 (0.9-1.6)	.204	1.1 (0.8-1.6)	.435
Unknown	0	6 (0.2)	N/A		N/A	
Birth presentation						
Cephalic	695 (94.2)	2646 (94.2)	1.0		1.0	
Breech	23 (3.1)	66 (2.4)	1.3 (0.8-2.2)	.236	1.2 (0.7-2.0)	.536
Other	20 (2.7)	98 (3.5)	0.8 (0.5-1.3)	.294	0.8 (0.4-1.4)	.379
Neonatal treatment						
Normal	665 (90.1)	2583 (91.9)	1.0		1.0	
Monitoring/NICU	73 (9.9)	227 (8.1)	1.3 (0.95-1.7)	.104	1.2 (0.9-1.6)	.210
Maternal high blood pressure						
No	699 (94.7)	2694 (95.9)	1.0			
Yes	39 (5.3)	116 (4.1)	1.3 (0.9-1.9)	.189	1.2 (0.8-1.8)	.384
Parity						
0	414 (56.1)	1150 (41.0)	1.7 (1.4-2.0)	<.001	1.8 (1.5-2.2)	<.001
1-2	296 (40.1)	1406 (50.1)	1.0		1.0	
≥3	28 (3.8)	248 (8.8)	0.5 (0.3-0.8)	.002	0.5 (0.3-0.7)	.001

AGA, appropriate for gestational age; *LGA*, large for gestational age; *NICU*, neonatal intensive care unit, *N/A*, not applicable; *SGA*, small for gestational age.
*Adjusted with maternal age, maternal SES, maternal psychiatric history, paternal age, and paternal psychiatric history.

have both been associated with both nulliparity[47,48] and Tourette syndrome.[24] In addition, changes in maternal hormonal environment, including higher testosterone levels in the first pregnancy, have been associated with parity.[49] The role of neuroendocrine mechanisms and Tourette syndrome is understudied, though there is a hypothesis, based on indirect measures, that patients with Tourette syndrome are exposed to higher concentrations of androgens in utero.[50] It has been suggested that autism in the child might lead to reproductive stoppage in the family, leading to reproductive choice biasing the relationship between parity and autism.[51] However, in our study the sibship size was added as a covariate, and Tourette syndrome is also likely to be diagnosed later than autism.[26,52]

Birth weight of 4000-4499 g was associated with decreased odds for Tourette syndrome. In general, the infant's size is an indicator of prenatal well-being, and both low and high birth weights have been associated with adverse long-term consequences such as obesity, metabolic disorders, and neurodevelopmental disorders.[53] Therefore, our finding was unexpected and intriguing. Some explanations associated with factors affecting birth weight can be hypothesized. Birth size is mediated by both genetic[54] and environmental factors, such as parity, maternal size, medication, smoking, and nutrition.[55] Thus, our finding raises the question of whether some of the factors associated with high birth weight (genetic or maternal size or nutrition), could have protective effects for Tourette syndrome. Replication of our findings and further studies examining the possible mechanisms are desirable to confirm the results and understand nature of the found association.

Low birth weight, gestational age, weight for gestational age, Apgar score at 1 minute, induced labor, birth presentation or birth type, neonatal treatment, or maternal blood

Table II. The final model including the factors associated with Tourette syndrome and covariates

	Cases	Controls	Final model*	
	n (%)	n (%)	OR (95%CI)	*P*
Birth weight (g)				
<1500	6 (0.8)	16 (0.6)	0.3 (0.02-3.9)	.334
1500-2499	26 (3.5)	68 (2.4)	1.1 (0.6-2.0)	.774
2500-3999	588 (79.7)	2074 (73.8)	1.0	
4000-4499	101 (13.7)	527 (18.8)	0.7 (0.5-0.9)	.013
≥4500	17 (2.3)	125 (4.5)	0.7 (0.4-1.3)	.255
Birth type				
Vaginal cephalic	547 (74.1)	2173 (77.3)	1.0	
Vacuum extractor/forceps/vaginal breech	63 (8.5)	178 (6.3)	1.1 (0.8-1.6)	.443
Planned cesarean	58 (7.9)	220 (7.8)	0.9 (0.6-1.2)	.378
Emergency/urgent cesarean	70 (9.5)	233 (8.3)	1.0 (0.7-1.2)	.794
Unknown	0	6 (0.2)	N/A	.977
Parity				
0	414 (56.1)	1150 (41.0)	1.7 (1.4-2.2)	<.001
1-2	296 (40.1)	1406 (50.1)	1.0	
≥3	28 (3.8)	248 (8.8)	0.5 (0.3-0.9)	.018

*Adjusted with maternal age, maternal SES, maternal psychiatric history, paternal age, paternal psychiatric history, the number of children in the sibship, and gestational age.

pressure were not associated with Tourette syndrome. Interestingly, associations between low birth weight and ADHD and autism, respectively, have been shown in several studies.[6-8] Furthermore, small for gestational age, low Apgar score, and prematurity have also been associated with both disorders and abnormal presentation, maternal high blood pressure, and neonatal treatment have been associated with autism.[6-9,11] As the examined factors can be considered as possible markers of several prenatal risk factors including intrauterine growth restriction, fetal distress, placental pathology, and pre-eclampsia, our results suggest that these adversities that may be involved in the etiology of ADHD or ASD, are not associated with Tourette syndrome. However, although this is the largest study of prospectively collected prenatal/perinatal risk factors for Tourette syndrome, the sample size is still somewhat small compared with sample sizes in prenatal epidemiologic studies about ADHD and ASD.[6-9] Therefore, the absence of the associations between some of the obstetric and neonatal factors and Tourette syndrome could potentially be due to lack of power.

The register-based design provides strengths such as prospectively collected data, a large, nationwide sample, data on several potential confounding factors, and validated diagnoses. However, not all children in Finland with Tourette syndrome are diagnosed and treated by specialist services[26] and, therefore, cannot be found in the FHDR. Nevertheless, virtually all children in Finland attend regular health screens at child health and school clinics. Thus, it is likely that the most severe cases of Tourette syndrome are recognized, referred to specialized health clinics, and registered. Another limitation is that although several maternal confounding factors could be controlled in the study, there are factors such as maternal nutrition and drinking behavior that are not available in the registers. Furthermore, Apgar score at 5 minutes would have been a better indicator of long-term outcomes in child than Apgar at 1 minute.[56] However, Apgar score at 5 minutes was not recorded in the FMBR during the whole study period. It was available only for 1% of the sample, and thus, it could not be chosen. In addition, the examined exposures, particularly the lowest birth weight and gestational age, were relatively rare. Thus, stratifying the cases based on common comorbidities including ADHD, ASD, and obsessive compulsive disorder would have required even larger sample size. No corrections of the *P* values were conducted for multiple comparisons; this can be accounted for while interpreting the results. However, correcting *P* values has not been a standard method in studies using epidemiologic categorized data and conditional logistic regression analyses.[7-9,12,13,33,34,41]

In conclusion, previously unrecognized factors as high birth weight and 3 or more previous births were associated with decreased odds for Tourette syndrome. In addition to examining the risk factors, identification of protective factors for Tourette syndrome may lead to better understanding of the etiology of the disorder. Further examination of the factors associated with prenatal growth or parity (eg, prenatal maternal nutrition, maternal hormonal levels during pregnancy, exposure to infections, or genetic factors) and their relationship with Tourette syndrome could help to clarify the nature of the detected associations. ■

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50 Years Ago in *The Journal of Pediatrics*

Charts of Normal Body Measurement and Revised Width-Weight Tables in Graphic Form

Pryor H. J Pediatr 1966;68:615-31

In 1966, Helen Pryor described the use of charts to aid parental understanding of their child's growth. Although Pryor used some variables and terms that are no longer used to define childhood growth (eg, "big boned" vs "small boned"), the challenge she described exists to this day. Parents get easily confused by the terminology we readily use as medical providers. In the study by Pryor, using charts and sharing a graphical description of growth points was found to improve parental understanding of childhood growth.

Numerous studies show that parents do not recognize when their children are above ideal body weight. Several challenges exist with using body mass index percentile (BMI%) even though it is the standard for discussing weight status with parents. Body composition, unexpected effects of greater height, and BMI rebound at age 4- to 6 years can make BMI% interpretation confusing for healthcare providers and parents. In an era when higher percentiles identify academic and athletic prowess, high BMI% may sound desirable to parents. Additionally, medical providers use BMI% to define the terms obese and overweight, but these terms have the potential to be off-putting or offensive to parents when not put into a clear medical context. Color-coded BMI charts to identify children in green (BMI% 5%-85%), yellow (BMI% 85%-95%), and red (BMI% >95%) zones have been shown as one way to enhance parent understanding of weight status.[1] Other strategies are also being employed or are under development.

Whether BMI% remains the vernacular for discussing childhood weight status or whether it goes the way of "big boned" remains to be seen. Regardless, if we are to engage parents and other caregivers to meet the challenge of this nation's pediatric obesity crisis, it is necessary for medical providers to use terminology and techniques that empower them and avoid those that stigmatize or confuse them.

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